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THE PROBLEM OF THE GENESIS OF ANEURYSMS AND ANGIOMAS OF THE BRAIN

[Following is the translation of an article by M. B. Kopylov entitled K Voprosu o Geneze Anevrizm i Angiom Golovnogo Mozga, (English version above), in Vopros Nevrokhirurgii (Problems of Neurosurgery), Vol. XXIV, No. 3, 1960, Moscow, pages 37-44.]

Distinctive aneurysmal or angiomatous vascular structures of the brain have now for a long time attracted the attention of pathologists (since 1761) and subsequently also that of neurosurgeons. This topic has been extensively developed following the introduction of the angiographic method.

In the literature (Padget, Cushing and Bailey, Murphy, Olivecrona and Ladenheim, Lindgren, Bremer and others) the extensive group of such vascular structures of the brain as arteriovenous aneurysms of the cavernous sinus, arterial aneurysms of the circle of Willis and angioma-like structures of the hemispheres are considered anomalies of embryonic development or as structures which develop against the background of a congenital insufficiency of various layers of the vessel wall. According to the composite data of Hamby, trauma is noted in only one to seven percent of the cases of aneurysm. This group includes only a disturbance in the integrity of the blood vessels by fractures of bones and by fissures at the base of the skull, as well as metal fragments, that is, a

disturbance of the blood vessel wall from without. Hamby presents other causes for these formations of aneurysms: infectious -- mycotic and luetic, hypertensive or arteriosclerotic, unclassified, etc. Angiomatous structures are regarded only as congenital anomalies. The absence of capillaries is explained by an aplasia of these blood vessels during the embryonic period.

In morphological works note is made of elements of hypoplasia of the muscle layer in the walls of aneurysmal blood vessels or insufficiency of the elastic membrane, which, in the opinion of a number of authors (Forbus, Richardson and Hyland and others), is the cause of dilatation and rupture of the blood vessel walls. Some morphologists note difficulty in differentiation and in making genetic conclusions from microscopic data at the late periods of development of the vascular structures (A. P. Avtsyn).

From the embryonic point of view it seems incomprehensible to us why the vascular pathology under analysis has its preferential localization in some areas and it is completely absent in others. Based on embryological premises the development of aneurysms might have been expected in many other areas where similar conditions of embryological development exist. Nor are we satisfied with the explanation of the absence of capillaries in angiomas as the hypoplasia of these blood vessels during the embryonic period. From embryological standpoints we cannot

understand the constant presence of direct arteriovenous connections in the arteriovenous aneurysms, angiomas, either.

The explanation of the frequency of development of arteriovenous aneurysms in the cavernous sinus and of arterial aneurysms near the circle of Willis has little basis from embryological standpoints, because we have not found any morphological data in the works of authors which, in the great majority of cases, should have confirmed the presence of developmental anomalies microscopically.

The ~~i~~ aim of the present work is to review the problem of the pathogenesis of vascular pathology considered congenital anomalies of vascular development in the literature.

We believe that the vascular changes are not the result of an embryonic developmental anomaly of the blood vessels but rather are acquired in connection with trauma, chiefly internal, as a result of momentary increase in the blood pressure in a section of the blood vessel wall at the time of skull trauma.

In order to discover the significance of the traumatic hemodynamic factor in the complicated combination of vascularization phenomena, we shall begin the study of it with those forms in which the role of the traumatic factor in the development of such structures is indubitable, that is, with aneurysms of the circle of Willis and with arterial and arteriovenous aneurysms in the cavernous sinus.

Blood is a liquid medium with a rather high [illegible] viscosity and is subordinate to the physical laws of hydraulics for a fluid which is contained in closed communicating [illegible] vessels -- it is incompressible, but it transmits pressure. The figures which express the blood pressure from an impact at the time it is administered from without at an unusuall high rate of speed and with consideration of the countering wave of blood, can increase momentarily to an unusual degree, impairing the normal maximum resistance of the blood vessel wall. We characterize such an effect of a blood wave on the blood vessel wall as a hydraulic shock (the theory of this shock has been worked out by N. Ye. Zhukovskiy).

The effect of the increased blood pressure on the blood vessel wall at the places of greatest curvature of it or at the place where a blood vessel comes off a main trunk at the greatest angle is certainly most destructive. Therefore, a considerable part of the aneurysms are formed at the abrupt curvatures in the "siphon" of the internal carotid artery or at the places where the arterial branches come off the main trunk.

The composite Tables (Fig. 1) presented by MacDonald and Korb and others, however, indicate that the maximum number of aneurysms occurs not so much at the terminal or middle portions of the blood vessels as at some sections determinable by other factors. It is possible that these places coincide with the points of maximum increase in fluid pressure against the blood vessel wall at the time of the hydraulic shock.

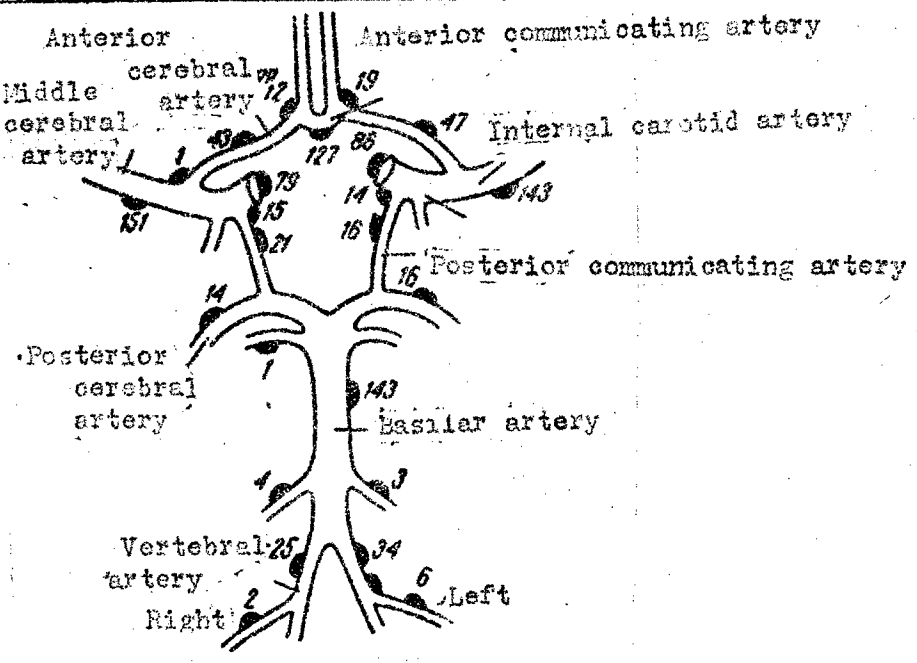

Fig. 1. Diagram of Localizations in 1023 Cases of Aneurysm (after MacDonald and Korb, 1939).

The absence of dampening of the hemodynamic shock under conditions of fixed, poorly ~~movable~~ movable blood vessels increases the possibility of their rupture. We explain the frequency of arteriovenous aneurysms in the cavernous sinus and the relatively small number of blood-vessel ruptures of blood vessels which are well protected by a fluid protective sleeve in the spinal-fluid cisterns.

The frequency of aneurysms in the blood vessels of the circle of Willis (see Fig. 1) is explained, apart from the considerations mentioned, also by disturbances in the blood flow, which we characterize as "conditions of an unstable equilibrium". Such conditions exist in the arterial and venous blood vessels, where the direction of the

blood flow is inconstant, and where the blood may flow in one direction or the other, which is determined by the physiological requirement and the difference in pressure in the blood vessels connected with these communicating blood vessels.

Under these "conditions of unstable equilibrium" the possibility of occurrence of a sudden wave of blood during skull trauma with a possible countering blood flow from the opposite side produces a momentary and extraordinary shock along the wall of the communicating vessel with distention or rupture of it. The development of such conditions during trauma is seen in arteries of the circle of Willis, in the end arteries of the brain, in the communicating venous sinuses and in the veins, particularly in the cavernous sinus.

It is perfectly understandable that the mechanical effect of the blood wave is more destructive at places where the blood vessel wall is weak, which situation is brought about either because of imperfections of its anlagen during the embryonic period or in connection with developmental anomalies of it subsequently, as well as in the case of reduced mechanical resistance of it in the event of infectious and metabolic degenerative processes in it.

Let us proceed with the explanation of the frequency of occurrence of these structures in the area which is defined by many authors as the area of the Rolandic fissures. From our point of view, it is

incorrect to define this area anatomically by landmarks.. The area of the great majority of the structures in which we are interested is so, not because of the Rolandic gyri but rather because of the extensive area of lateral surface of the brain hemispheres and of the skull, where the greatest frequency of aneurysms is observed.

Since the time of the introduction of arteriography, cerebral angiology has been expanded extraordinarily. Tremendous material has been accumulated and everyone occupied in cerebral angiography knows how variable the blood vessels -- the arteries and particularly the veins in the lateral portions of the skull and brain at all levels of the circulation -- are in the bones and in the dura and pia maters. While in the sagittal planes both the arteries and the veins constitute relatively constant collecting channels, in the lateral portions the limits of variation are much wider, particularly at the venous stage of the circulation. The sagittal sinuses and veins are relatively stable; however, the development of the venous blood vessels in the parietotemporal portions of the vault are so individual that it is hardly possible to find two parietal areas with similar vascularization among an abundance of angiographic material (we repeat: this is observed at various circulatory levels).

Apart from the existence of a multitude of communications between large vessels, characterized by an inconstant equilibrium of the blood

flow, the same conditions apparently obtain even in the small terminal vessels and in the capillaries. Naturally, the possibility of marked pressure rises in these terminal vessels must be supposed at the time of an unexpected and considerable acceleration in the blood flow from a shock through the closed, completely incompressible, hard housing of them -- the skull.

As a consequence of trauma obliteration of part of the capillaries has been noted by a number of authors (B. N. Klosovskiy and others). The remaining intact blood vessels of this area have to carry the same quantity of blood but at an even greater rate and higher pressure, which, in our opinion, also leads to a reorganization of their walls and a conversion of capillaries into larger blood vessels.

These considerations permit us readily to explain the existence of such frequently occurring direct arteriovenous connections in the structures in which we are interested from a hydrodynamic point of view.

Afterwards, the post-traumatic blood vessel disturbances may give rise to the necessity for an unusual development of blood vessels and communications of a compensatory nature, which also leads to the formation of vascular forms, to select a name for which is difficult for all authors and which in some cases may be called "aneurysms"; in others, "arteriovenous aneurysms" or "arteriovenous anastomoses"; and in still others, "arteriovenous angiomas", etc. These forms are post-traumatic

compensatory vascular structures, and only part of them are congenital.

In 72 of our observations on carotid-cavernous-sinus aneurysms, there were fractures and fissures of the base of the skull in 30 percent, and an impairment of the blood vessel wall from without may be suspected. With the exception of solitary instances of infectious etiology, in all the other observations of carotid-cavernous-sinus aneurysms the direct cause was closed trauma of the skull, where it was necessary to consider that the wall of the carotid artery had been impaired from within under the influence of the hydraulic shock (Fig. 2, a, b, c, d).

Fig. 2. Two Carotid-Cavernous-Sinus Aneurysms After Closed Traumas of the Skull with the Blood Flow Which Developed.

a,b--anteriorly, along the orbital veins; c,d--posteriorly along the basilar veins.

Therefore, the chief significance of the hydrodynamic factor in the impairment of the blood vessel wall is indubitable as far as we are concerned, and this factor represents specifically the influence of the hydraulic shock both in tears of the arterial wall in the cavernous sinus and in aneurysms of the area of the circle of Willis.

Proceeding with the analysis of 38 cases (there was trauma in the history of 12 of them), of so-called "arteriovenous aneurysms" or "angiomas", we divide them into several groups.

In the first group we include lesions which penetrate deeply into the brain tissue, sometimes not only reaching the midline but also the opposite side. Morphological descriptions of lesions of this group indicate gross abnormalities in the brain tissue of dysembryogenetic nature (Lindgren, Cushing). The lesions of this group undoubtedly are congenital and should be included among the malformations.

By X-rays of the skull and contrast methods an extension of the disturbances over a number of layers is frequently found in this case. Changes may be found in the bones, in the blood vessels of the dura mater and in the cerebral tissue at the time of pneumoencephalography (a focal enlargement of the general brain mass with a displacement and compression of the ventricles or a reduction of its mass and a regional dilatation of the spinal fluid spaces).

The second group is characterized by features in the

development only of the pial blood vessel. Changes in the brain tissue may be absent, and if they have developed they are of secondary character and are expressed as softenings or atrophic-cicatricial changes in connection with an inadequacy of vascularization of brain tissue for a long period of time. Angiographically a group of uniformly or non-uniformly dilated blood vessels is seen. According to the data of morphologists, the absence of capillaries is characteristic of the lesions of this group. We believe that a group of such blood vessels develops from capillaries in connection with a dilatation of them.

This group of vascular pathology can be referred formally to the group of angiomas and is probably the consequence of the improper development of blood vessels during the embryonic period. While adhering to this viewpoint and giving due importance to the defects in the embryonic anlagen, we do not consider all the cases of this group congenital, however, and we do not attribute all of them to defects in the development of their walls. We believe that this tangle of blood vessels should be considered as being formed from capillaries which have been dilated because of hemodynamic factors.

In the second group of malformations we include cases where the existence of a large number of blood vessels is connected with a compensatory development of them for the purpose of filling in a congenital insufficiency -- hypoplasia or aplasia of various large blood vessels

or sinuses. Changes of this kind are seen chiefly in the venous system of cerebral blood vessels. However, even in this group of disturbances we cannot always confidently prove the existence of congenital hypoplasia or aplasia of the blood vessel by the methods of intravital examination and exclude the possibility of a disturbance in its patency associated with impairment of its walls and in the lumen by diseases of acquired nature, of infectious etiology or other genesis. Marked dilatations of the internal cerebral vein and of branches coming into the sagittal plane of a compensatory nature (veins of the septum pellucidum) speak for insufficiency, apparently congenital, of the venous collectors of the mid-line: the inferior sagittal sinus, in spite of its being common, cannot be seen on any angiogram of the cases under analysis (Fig. 3).

In the third group of developmental anomalies we include cases where there is no congenital anomaly in the vascular pathology or where only a local insufficiency of the wall associated with the formation of this blood vessel is possible. In this group we include those forms of vascular pathology of the brain in the formation of which, from our point of view, the main part is played by trauma.

Such forms should have been called "arterial, arteriovenous or venous aneurysms". They cannot be considered as belonging to the groups of angiomas, as has been done by some authors (Fig. 4). We have at our disposal a small number of autopsy cases of this group, but, naturally, we have

no data of angiographic examinations prior to trauma for the purpose of comparison with post-traumatic pictures. These circumstances complicate the proofs.

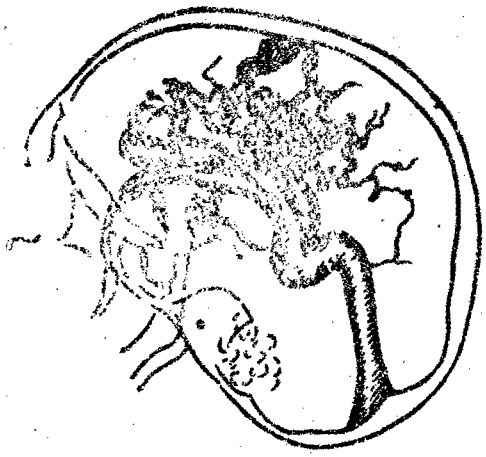

Fig. 3. Congenital Anomaly of Development of Internal Cerebral Vein with Hypoplasia of the Inferior Sagittal Sinus

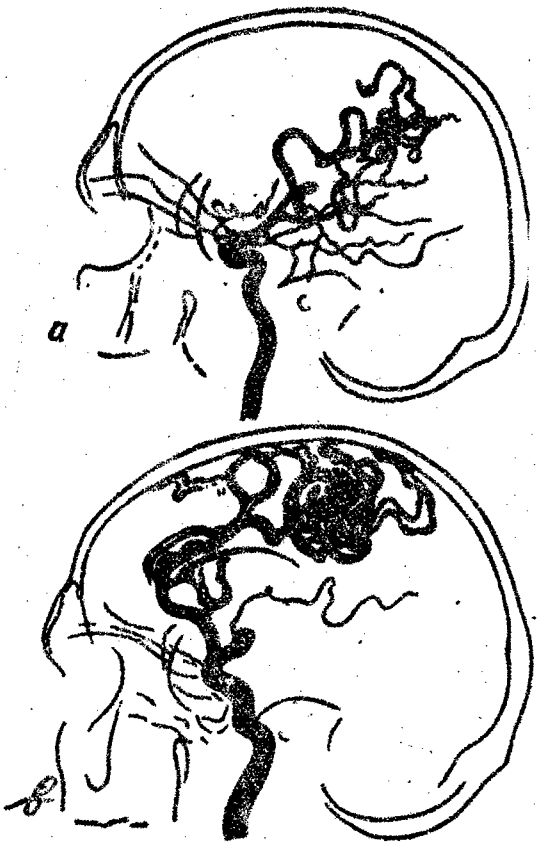

Fig. 4. Arteriovenous Aneurysms on both Sides--on the Anterior Cerebral Artery on the Right (a) and on the Middle Cerebral on the Left (b). The Anterior cerebral artery on the left and the middle cerebral on the right have been obliterated. There was a history of cerebral contusion.

Let us summarize what has been said. The viewpoint of the genesis of the so-called "arteriovenous aneurysms -- angiomas" which has been established in the literature as being developmental anomalies

is without adequate basis, we believe.

We recognize the part of dysembryogenesis only in part of the cases.

The embryological theory /leaves/ a number of factors unclarified -- the frequency of impairment of the blood vessel walls at certain levels: a) in the cavernous sinus; b) at the level of the circle of Willis and c) in the area of the Rolandic fissures.

The explanation of the absence of capillaries in arteriovenous aneurysms -- angiomas -- is unconvincing.

The frequency of direct arteriovenous anastomoses in the lesions under analysis is incomprehensible.

Based on the embryological theory, the existence of anomalies in other portions of the central nervous system might have been expected.

Giving what is due to the embryological factors, we ascribe great importance to functional hemodynamic factors in the origin of aneurysms and arteriovenous aneurysms -- angiomas.

By the mechanical hemodynamic factors of a momentary, marked rise in blood pressure on the blood vessel wall during skull trauma -- the hydraulic shock -- light is thrown on a number of insufficiently or completely unexplained problems in the origin of both arteriovenous and arterial aneurysms.

The lumina of arterial and venous blood vessels of the brain

and skull do not have any valves to interfere with the flow of blood. Pressure on the blood vessel wall is proportional to the square of the rate of movement of the fluid along the blood vessel, by which the dilatation and reorganization of the capillary, arteriole and venule walls is explained in the angiomatous lesions. In addition to this, in the presence of the hard incompressible protective frame around the brain -- the skull -- the force of the shock through the skull is largely taken up by fluids in the spinal-fluid spaces of the brain as well as by the blood in its blood vessels. Momentarily, unusually large pressures are added to the ordinary blood pressure, and the blood pressure against the blood vessel wall acquires the significance of a hydraulic shock in it. In the case of a counter-wave of blood the site of maximum hydraulic pressure against the blood vessel wall is determined by the point at which summation of both waves occurs (formula of N. Ye. Zhukovskiy).

In the event of a curve in the blood vessel the greatest pressure is taken up by the inside of the external arc of the blood vessel and this is more marked the more abrupt the curve in the blood vessel, and in the event that it branches, it is more marked the greater the angle at which the branch leaves the main trunk.

The sharp jump in the blood pressure in the cerebral vessels which occurs in the case of head trauma, particularly closed trauma is related not only to the existence of the hard frame of the skull

but also to the degree of fixation of the blood vessel itself or part of it in the skull. Therefore, the influence of the hydraulic shock on the wall of the internal carotid artery which has been fixed at the cavernous sinus, should exert its influence, and specifically here so frequently leads to the formation of arteriovenous anastomoses even without any embryonic defect in the blood vessel. The existence of embryonic defects and of embryonic vascular remnants (Krayenbühl and Yasargil) increases the number of such ruptures only to a certain degree. The method of angiography demonstrates the preservation of these embryonic blood vessels with exceptional rarity.

The existence of a large percentage of aneurysms of the internal carotid artery as it leaves the cavernous sinus in the vicinity of its place of division as well as at the beginning of the anterior and middle cerebral arteries is determined to a considerable degree by the great curvatures of the blood vessels here; the large number of aneurysms in the blood vessels of the circle of Willis, by the presence of conditions of an unstable equilibrium in the hemodynamics here. The dilatation of the small terminal blood vessels forming the arteriovenous aneurysms -- angiomas -- in the cerebral hemispheres is determined by these conditions as well as by the involvement of part of the capillaries in the trauma.

The predominance of their number in the lateral portions of the hemispheres is determined, in our opinion, by the great variability of

the blood vessels, that is, if we can express ourselves in this way, by the great instability of the forms of development of the blood vessels here during the course of ontogeny.

Among the fixed blood vessels ruptures of the superior longitudinal and transverse sinuses from closed trauma without fractures of the bones are extremely rare in adults. Here, the successive reduction in the rate of movement of the blood wave by the transverse septa -- projections on the inner surface of these sinuses described by K. D. Balyasov -- lessens the influence of the hydrodynamic factor.

These structures, in our opinion, are of physiological importance as devices which direct and check the flow of blood at times of excessive accelerations of it; devices which prevent the harmful influence of the hydraulic shock. Among these dampening devices which lessen the harmful influence of skull trauma on the cerebral vessels generally and of increased pressures of blood in the vessels in it in particular mention should be made of the fact that the blood vessels are surrounded by repositories of a movable fluid medium -- the spinal-fluid cisterns.

The influence of the neuroregulatory factor at the time of marked increases in the blood pressure should be considered spastic; in some of the cases ultraboundary stimulation may produce no reaction. With either result the hydraulic shock may exert its destructive effect on the integrity of the blood vessel wall.

In conclusion, we should like to note that the existence of infectious foci, the influence of [illegible] intoxication, of metabolic-degenerative changes, the influence of both exogenous and particularly endogenous trauma in the form of the momentary and excessive rise in blood pressure during skull trauma which we have noted -- all these factors which destroy the walls of cerebral blood vessels are responsible for their impairment.

The factors of congenital deficiency of cerebral blood vessels are not of such great significance as they are considered to be in the literature.

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